Adherence to a Ketogenic Low-Carbohydrate, High-Fat Diet Is Associated With Diminished Training Quality in Elite Racewalkers

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Purpose: To examine the effects of a high-carbohydrate diet (HCHO), periodized-carbohydrate (CHO) diet (PCHO), and ketogenic low-CHO high-fat diet (LCHF) on training capacity. Methods: Elite male racewalkers completed 3 weeks of periodic training while adhering to their dietary intervention. Twenty-nine data sets were collected from 21 athletes. Each week, 6 mandatory training sessions were completed, with additional sessions performed at the athlete’s discretion. Mandatory sessions included an interval session (10 × 1-km efforts on a 6-min cycle), tempo session (14 km with a 450-m elevation gain), 2 long walks (25–40 km), and 2 easy walks (8–12 km) where “sleep-low” and “train-low” dietary strategies were employed for PCHO. Racewalking speed, heart rate, rating of perceived exhaustion, and blood metabolites were collected around key sessions. Results: LCHF covered less total distance than HCHO and PCHO (P < .001); however, no differences in training load between groups were evident (P = .285). During the interval sessions, walking speed was slower in LCHF (P = .001), equating to a 2.8% and 5.6% faster speed in HCHO and PCHO, respectively. LCHF was also 3.2% slower in completing the tempo session than HCHO and PCHO (P = .001). Heart rate was higher (P = .002) and lactate concentrations were lower (P < .001) in LCHF compared to other groups, despite slower walking speeds during the interval session. No between-groups differences in rating of perceived exhaustion were evident (P = .077). Conclusion: Athletes adhering to an LCHF diet showed impaired training capacity relative to their high-CHO-supported counterparts, completing lower training volumes at slower speeds, with higher heart rates.

Keywords: ketones, periodized, lactate, endurance training, CHO availability, athlete

Knowledge and practice around the ideal training diet for endurance athletes has evolved from universally high carbohydrate (CHO) intakes to consistent high CHO (HCHO) availability (matching CHO intake to the acute fuel demands of each training session) and, more recently, to periodized CHO (PCHO) availability (high availability for training quality and low availability to increase metabolic adaptation, according to the characteristics of each session). However, the last decade has seen renewed interest in ketogenic, low-CHO, high-fat (LCHF) diets as an alternative strategy to enhance endurance performance via increased reliance on a more abundant muscle fuel source. Our investigation of the effect of these different approaches to the training diet on exercise metabolism and performance in elite endurance athletes found that although the LCHF diet achieved a doubling of fat oxidation, it resulted in an impairment of real-world sport performance compared to diets providing HCHO or PCHO availability. In the current paper, we summarize the effects of these diets on training capacity.

Methods

Participants

Twenty-one male racewalking athletes (27 [4] y, 65.8 [7.3] kg, 64.3 [5.8] mL·kg⁻¹·min⁻¹ VO₂max) participated in this study, which involved 2 research-embedded training camps held at the Australian Institute of Sport in November 2015 and January 2016. Eight of these athletes completed both training camps, resulting in 29 data sets. Participants included tier 5 (eg, Olympic and World Championship medalists, national and continental record holders) and tier 4 athletes (eg, junior international athletes and training partners of world class athletes). Athletes were educated about the potential benefits and limitations of each diet before being surveyed about their preferred intervention. Noting dietary beliefs and athletic characteristics (ie, age, body mass, VO₂max, personal bests, training history, and intended training load during the camp), we allocated athletes into HCHO diet (n = 9), PCHO (n = 10), and LCHF (n = 10) groups. We note that athletes who participated in both training camps were assigned to a different dietary intervention on the subsequent occasion with care to equalize the crossover to the second diet as well as possible (eg, 2–3 athletes).

Study Overview

This study conformed to the standards of the Declaration of Helsinki and was approved by the Australian Institute of Sport Human Research Ethics Committee (#20150802). All participants provided written informed consent after receiving detailed descriptions of the protocols.

Between 2 test batteries, athletes undertook a 3-week training program that included 6 mandatory racewalking sessions per week (Figure 1) and additional self-chosen sessions of racewalking and cross-training (swimming, running, cycling, and resistance training). The mandatory sessions, integrated into the weekly program to periodize overall training volume and appropriately stress each energy system, were:
Figure 1 — Mandatory weekly training sessions completed by athletes adhering to the HCHO, PCHO, and LCHF diets. H and L represent high and low CHO support during training. Daily training volume (in kilometer) is shown for week 2 for each dietary group, to show the periodization that occurred over the week, and was generally similar between dietary groups. CHO indicates carbohydrate; HCHO, high-CHO diet; LCHF, ketogenic low-CHO high-fat diet; PCHO, periodized-CHO diet.
1. Interval training—weekly session (10 × 1 km on 6-min cycle) undertaken on a 400-m athletics track.
2. Tempo (hill) session—weekly road session involving 14-km distance with a total elevation gain of ~450 m.
3. PCHO sessions—athletes in the PCHO group undertook 2 sessions/wk, each involving 8 to 12 km at an easy pace, undertaken with low CHO availability.
   a. A “train low” model\(^1\) implemented as an afternoon session, using the morning tempo session to deplete muscle glycogen, then restricting dietary CHO at intervening meals to expose the next workout to low endogenous and exogenous CHO availability.
   b. A “sleep-low” model\(^1\) implemented as a morning fast session, using the previous afternoon’s interval session to deplete muscle glycogen stores, then restricting CHO in the evening meal.

HCHO and LCHF groups also completed both sessions, with high and low CHO support, respectively.
4. Long walks—25 to 40 km undertaken twice per week on a flat route. Nutrition support, consistent with each athlete’s dietary group provided every ~2 km on the route.

Before and after each session, fingertip capillary blood samples were collected to measure blood lactate (Lactate Pro 2, Arkray), as well as ketone bodies (β-hydroxybutyrate [βHB]) and glucose concentrations using the FreeStyle Optium Neo device (Abbott Diabetes Care). Time to complete each repetition of the interval sessions, and the hill tempo course were recorded and used to calculate average walking speed, reported as an absolute walking speed (in kilometer per hour) and as a percentage of the athlete’s velocity at VO\(_2\)\(_{\text{max}}\) (\(\nu\)VO\(_2\)\(_{\text{max}}\)). During the interval session, heart-rate (HR) monitors (Forerunner, Garmin International) recorded average heart rate, and session ratings of perceived exertion (RPE) were recorded (6–20, Borg Scale). Ratios of both HR and RPE (as internal load measures) relative to average walking speed were calculated to produce an “exertion/velocity ratio.”\(^6\) An increase in these metrics for any given speed suggests that the session was more demanding, physiologically (HR) or perceptually (RPE). Athletes completed an electronic training diary to document all training (both mandatory and self-selected sessions), including total volume (in kilometers), exercise duration (in minutes), and a RPE (0–10). Session RPE, calculated by multiplying active minutes by RPE,\(^7\) was used as a proxy measure of weekly training load.

**Dietary Control**

Athletes were housed at the Australian Institute of Sport residence where all food and fluids consumed during the study were created, prepared, and monitored by a chef, and food service/sports dietitians within the research team:\(^9\)
1. HCHO\(^1\): 60% to 65% energy from CHO, 15% to 20% protein, 20% fat, with CHO intake being similar across days and consumed before, during, and after all training sessions.
2. PCHO\(^1\): Energy/macronutrient matched to HCHO, but distributed differently between and within days, to achieve some training sessions with high CHO availability (high muscle glycogen, CHO feeding during session) and others with low CHO availability (low preexercise glycogen, overnight fasted or delayed postsession refueling).
3. LCHF: Energy matched to HCHO but <50 g/day CHO, 75% to 80% of total energy from fat, and 15% to 20% protein. Based on the ketogenic LCHF diet popularized by a lay book written by exercise scientists and featuring menus and recipes.\(^9\)

**Statistical Analysis**

One data set was removed due to unrelated training inconsistencies, leaving a total of 28 data sets (20 athletes) for analysis. All data were initially checked for normal distribution via visual inspection of QQ-plots and residual plots. Since significant deviations were apparent for lactate and glucose concentrations, data were log-transformed. Using R Studio, general linear mixed models were created using fixed effects of dietary group, week and, where required, time point. For all models, a random effect for participant identification and camp was included. Models were estimated using Restricted Maximum Likelihood. \(P\) values were obtained using type II Wald \(F\) tests with Kenward–Roger degrees of freedom. Where significant fixed effects were evident, Tukey post hoc comparisons were performed to identify specific condition differences. Cohen’s effect sizes were also calculated. Significance was set at \(P < .05\).

**Results**

Significant fixed effects for diet (\(P < .001\)) and week (\(P < .001\)) were evident for total training volume (Figure 2A). LCHF covered less total distance than HCHO (\(d = 0.35; \ P = .006\) and PCHO groups (\(d = 0.69; \ P < .001\)), and training distance in week 3 was lower than weeks 1 (\(d = 1.11; \ P < .001\)) and 2 (\(d = 1.27; \ P < .001\)) for all groups. Despite a lower training volume in LCHF, no differences in training load occurred between dietary groups (Figure 2B; \(P = .285\)).

**Interval Sessions**

During the interval sessions (Figure 3), both absolute walking speed (\(P = .001\)) and speed normalized to \(\nu\)VO\(_2\)\(_{\text{max}}\) (\(P < .001\)) were slower in LCHF, equating to a 2.8% and 5.6% faster absolute speed and 8.0% and 8.2% faster normalized speed in HCHO and PCHO, respectively. A training effect was evident for all groups (\(P < .001\)), with a faster absolute walking speed in weeks 2 (\(d = 0.44; \ P = .027\)) and 3 (\(d = 0.54; \ P = .001\)) than week 1. Normalized speed was faster in week 3 compared to week 1 (\(d = 0.56; \ P = .003\)). No interaction for either absolute (\(P = .093\)) or normalized speed was reported (\(P = .224\)), suggesting a similar rate of improvement across groups. Heart rate was higher in LCHF compared to HCHO (\(d = 0.61; \ P = .010\)) and PCHO (\(d = 1.07; \ P = .005\)), with a general decrease noted from week 1 to week 3 (\(d = 0.42; \ P = .009\)). There were no differences in RPE between groups (\(P = .077\)) or over time (\(P = .413\)). Both the HR/speed (\(P < .001\)) and RPE/speed (\(P < .001\)) ratios were elevated in LCHF compared to HCHO and PCHO. For all groups, a decrease in the HR/speed ratio occurred with each week (\(P < .001\)), with the RPE/speed ratio being lower at week 3 compared to week 1 (\(P = .036\)) only. Lactate concentrations increased postexercise (Table 1; \(P < .001\)); however, the increase was greater in HCHO (\(P = .002\)) and PCHO (\(P < .001\)) compared to LCHF. Blood glucose levels increased postexercise in the HCHO and PCHO groups (both \(P < .001\)), with no increase evident in the LCHF group (\(P = .292\)).
during any train-low/sleep-low session for all dietary conditions occurred in LCHF (+ 1.1 mM; walks, a small but signifi-

cant increase in postexercise lactate concentrations during the train-

sible changes for total training load (defined as training volume multiplied by session RPE), the LCHF group experienced a higher perceived exertion per kilometer travelled and a higher perceptual (RPE) and physiological (HR) effort for any given speed. The LCHF diet was associated with a sustained increase in concentra-

ions of blood β2HB concentrations above the level considered to signal ketosis (0.5 mM) and within proposed ideal zone for improvement of exercise performance (1–3 mM). We cannot rule out the possibility that the 3-week period provided inadequate time to allow the LCHF-supported athletes to adapt to the diet and restore training intensity and quality. However, when considering (1) the lack of difference in the rate of improvement in training quality between groups, (2) other data from this study that explain an ongoing metabolic limitation for sustained high-intensity exercise, and (3) preliminary evidence that selective CHO intake during high-intensity exercise enhances performance in chronically keto-adapted athletes, it appears that sustained LCHF does not provide the ideal nutritional support for high-performance endurance athletes who compete in events involving sustained or periodic expenditure at very high absolute and relative exercise intensities. Renewed interest in the ketogenic LCHF diet has led to prospective (1–12 wk) and cross-sectional investigations of the long-term (>6 mo) effect of this diet on exercise metabolism and performance (see review). However, the impact of such diets on training characteristics has not been well described. Our study implemented a pragmatic training program suitable to world-class athletes, where 6 sessions were prescribed and monitored within each weekly cycle, allowing athletes to add extra sessions to individualize the training program according to immediate and general training history and event specialization. The program was developed in collaboration with several tier 5 athletes and their coaches, and included appropriate sequencing of low-, medium-, and high-volume days (Figure 1) to facilitate adaptation and maximize performance. Overall training volume ranged between 112 and 138 km/wk for the first 2 weeks, before reducing to 97 to 107 km/wk in week 3. It is unclear whether the decrease in volume

Blood lactate, glucose, and ketone concentrations during the train-

low, sleep-low, and long walk sessions are presented in Supplementary Tables S1 and S2 (available online). During the long walks, a small but significant increase in postexercise lactate occurred in LCHF (+ 1.1 mM; P < .001), but did not change in HCHO (+ 0.6 mM; P = .989), PCHO (+ 0.2 mM; P = .998), or during any train-low/sleep-low session for all dietary conditions (P > .05). Blood glucose was lowest and ketone concentrations highest in LCHF compared to the other groups during all sessions (P < .05). Preexercise blood ketone concentrations (Figure 5) show a significant increase on day 3 in LCHF and remained > 0.5 mM throughout the camp (P < .001).

Adverse Reactions

Three LCHF athletes developed dermatitis, consistent with prurigo pigmentosa, within weeks 1 to 2 of the dietary change. This was manifested as dense collections of itchy hyperpigmented papules on the chest and back which rapidly resolved with reintroduction of CHO-rich foods at the study completion.

Discussions

We describe the characteristics of training undertaken by elite male racewalkers during a 3-week period in early season training, with 3 different approaches to dietary support: consistent high CHO availability, PCHO availability, and a ketogenic low-CHO high-fat diet. The rigor of dietary control and training monitoring in high-performance athletes across different dietary approaches provides novel and robust data. Although all groups increased their training quality (speed) over the training camp, training completed by the LCHF athletes was lower in both volume and intensity compared to HCHO and PCHO groups. In the absence of between-group differences for total training load (defined as training volume multiplied by session RPE), the LCHF group experienced a higher perceived exertion per kilometer travelled and a higher perceptual (RPE) and physiological (HR) effort for any given speed. The LCHF diet was associated with a sustained increase in concentra-

Figure 2 — (A) Total training volume (in kilometers) and (B) total training load (training duration multiplied by RPE) during week 1, week 2, and week 3 of the training camp for the HCHO, PCHO, and LCHF groups. Data shown as mean (SD). *Significantly lower than HCHO and PCHO. #Significantly lower than week 1 and week 2. HCHO indicates high-carbohydrate diet; LCHF, ketogenic low-carbohydrate high-fat diet; PCHO, periodized-carbohydrate diet; RPE, rating of perceived exhaustion.

Metabolites

For the tempo sessions (Figure 4), a significant fixed effect for diet was evident for both absolute (P < .001) and normalized walking speed (P < .001), with LCHF being 3.2% slower (~2.3 min) in completing the 14 km route than HCHO and PCHO. Postexercise lactate concentrations increased in all groups (P < .001), but were significantly lower in LCHF compared to HCHO and PCHO (P < .001) and PCHO (P < .001). Preexercise glucose concentrations were lower in LCHF compared to HCHO (P < .001) and PCHO (P < .001) and decreased following the session (P < .001), without changing in HCHO (P = .996) or PCHO (P = .716). Although there were no differences in ketone concentrations between groups during week 1, by weeks 2 and 3, they were significantly elevated in LCHF compared to HCHO and PCHO (Table 1; P < .001).
was an intentional measure to prepare for the 10,000-m race which followed the training block (4 d later) or represented a natural response to consecutive weeks of heavy training. Nevertheless, the incorporation of low-, medium-, and high-volume days within and between training weeks (Figure 1) was successful in facilitating a training adaptation, with a ∼3% to 7% increase in \( \dot{V}O_2\text{max} \) evident in all athletes.

Across the 3 weeks, the LCHF group consistently completed less training mileage than HCHO and PCHO. Because the camps took place at the commencement of the training season, we were unable to obtain reliable training data for the previous weeks to establish a true baseline training volume. Therefore, it is unknown whether the LCHF group had a true decline in training volume, or whether lower mileage was simply a characteristic of this group. However, our groups were well matched for personal best times, age/training age, and 20 versus 50 km specialists, although there was a trend for the LCHF group to have a higher \( \dot{V}O_2\text{max} \) and personal best times than the HCHO group. Therefore, we see no reason for inherently lower training mileage to occur in the LCHF group. In addition to reduced training volume, walking speed (both absolute and relative to \( \dot{V}O_2\text{max} \)) was significantly slower during both the interval and tempo training sessions in LCHF. Although

\[ \text{Figure 3} \] Weekly interval training session results for the athletes on HCHO, PCHO, and LCHF diets. (A) Average absolute rep speed, (B) average rep speed normalized to \( \dot{V}O_2\text{max} \), (C) average heart rate, (D) RPE, (E) heart-rate:speed ratio, and (F) RPE:speed ratio. Data shown as mean (SD) and individual results. *Significant difference compared to HCHO and PCHO. #Significant difference compared to week 1. ^Significant difference compared to week 2. HCHO indicates high-carbohydrate diet; LCHF, ketogenic low-carbohydrate high-fat diet; PCHO, periodized-carbohydrate diet; RPE, rating of perceived exhaustion.
Table 1  Blood Lactate, Glucose, and βHB Concentrations Preexercise and Postexercise During the Interval and Tempo Sessions on Week 1, Week 2, and Week 3 in the HCHO, PCHO, and LCHF Groups

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<td>Week 1</td>
<td>1.8 (0.6)</td>
<td>10.1 (5.2)*</td>
<td>5.1 (0.7)</td>
<td>7.2 (3.3)*</td>
<td>0.2 (0.1)</td>
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<td>Week 2</td>
<td>1.3 (0.3)</td>
<td>9.8 (1.7)*</td>
<td>5.2 (0.8)</td>
<td>7.4 (2.3)*</td>
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<td>Week 3</td>
<td>1.7 (0.6)</td>
<td>8.0 (3.6)*</td>
<td>4.5 (0.3)</td>
<td>6.5 (0.9)*</td>
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<td><strong>PCHO</strong></td>
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<td>Week 1</td>
<td>1.8 (0.5)</td>
<td>11.9 (4.9)*</td>
<td>5.5 (0.7)</td>
<td>7.9 (1.6)*</td>
<td>0.3 (0.1)</td>
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<td>Week 2</td>
<td>1.3 (0.3)</td>
<td>9.7 (4.4)*</td>
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<td>Week 3</td>
<td>1.4 (0.4)</td>
<td>10.3 (3.4)*</td>
<td>4.9 (0.4)</td>
<td>8.4 (3.2)*</td>
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<td>Week 1</td>
<td>1.3 (0.3)*</td>
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<td>4.7 (0.3)*</td>
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<td>Week 2</td>
<td>1.0 (0.1)*</td>
<td>7.6 (3.8)*,#</td>
<td>4.8 (0.3)$</td>
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<tr>
<td>Week 3</td>
<td>1.0 (0.3)*</td>
<td>6.5 (1.7)*,#</td>
<td>4.8 (0.4)</td>
<td>5.6 (0.7)</td>
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|                  |         |         |         |         |         |         |
| **Tempo**        |         |         |         |         |         |         |
| **HCHO**         |         |         |         |         |         |         |
| Week 1           | 2.2 (0.5)| 6.6 (2.2)*| 5.1 (1.0)| 5.4 (0.9)| 0.1 (0.1)| 0.2 (0.0)|
| Week 2           | 2.0 (0.8)| 6.3 (2.3)*| 5.8 (1.1)| 5.6 (1.3)| 0.2 (0.1)| 0.2 (0.0)|
| Week 3           | 2.1 (0.7)| 7.9 (3.7)*| 5.0 (0.9)| 5.2 (1.1)| 0.1 (0.0)| 0.2 (0.0)|
| **PCHO**         |         |         |         |         |         |         |
| Week 1           | 2.0 (0.8)| 6.8 (2.8)*| 5.3 (0.7)| 5.3 (1.0)| 0.1 (0.0)| 0.2 (0.1)|
| Week 2           | 2.8 (0.7)| 7.1 (2.0)*| 6.4 (1.4)| 5.2 (1.0)| 0.2 (0.1)| 0.2 (0.0)|
| Week 3           | 2.1 (0.4)| 9.3 (2.0)*| 6.3 (1.6)| 6.3 (1.3)| 0.1 (0.0)| 0.2 (0.0)|
| **LCHF**         |         |         |         |         |         |         |
| Week 1           | 1.9 (2.2)*| 5.2 (3.8)*,#| 4.0 (0.4)#| 2.8 (0.8)*,#| 0.2 (0.1)| 0.3 (0.2)|
| Week 2           | 1.1 (0.3)*| 4.6 (2.6)*,#| 4.3 (0.8)$ | 3.4 (0.9)*,# | 1.5 (0.9)$^,^| 1.3 (0.9)$^,^|
| Week 3           | 1.2 (0.7)*| 4.3 (1.5)*,#| 4.1 (0.5)$ | 4.0 (0.6)*,# | 1.5 (1.0)$^,^ | 1.1 (0.6)$^,^|

Abbreviations: HCHO, high-carbohydrate diet; LCHF, ketogenic low-carbohydrate high-fat diet; PCHO, periodized-carbohydrate diet.

*Significant increase from preexercise. #Significant difference compared to both the HCHO and PCHO groups. $Significant difference to PCHO only. *Significant difference to week 1.

Figure 4 — Weekly 14-km tempo training session results for the athletes on HCHO, PCHO, and LCHF diets. (A) Average absolute speed and (B) speed normalized to v̇VO$_2$max. Data shown as mean (SD) and individual results. *A fixed effect for diet, where LCHF is significantly slower than HCHO and PCHO. HCHO indicates high-carbohydrate diet; LCHF, ketogenic low-carbohydrate high-fat diet; PCHO, periodized-carbohydrate diet; v̇VO$_2$max, velocity at maximal oxygen uptake.
the LCHF group improved their outputs during the interval session over the course of the training camp, they still failed to reach the absolute or relative speeds achieved by the HCHO and PCHO athletes. Despite training at lower absolute and relative velocities in these sessions, LCHF athletes had elevated heart rates and similar RPEs to athletes on CHO-rich diets. When these metrics were expressed relative to training velocity, the exertion/velocity ratios of the LCHF group decreased over the training period but remained significantly higher than their CHO-supported counterparts, showing an apparent uncoupling of speed and both physiological (heart rate) and perceptual (RPE) effort. By day 3 of LCHF, blood βHB was sustained above concentrations considered to represent ketosis and within the range proposed as the ideal zone for improvement of exercise performance (1–3 mM). However, although ketone bodies can provide a substrate for peripheral tissues such as muscle, maximal rates of ketone-supported mitochondrial respiration are very low, particularly when competing with CHO or fat as a substrate. Instead, the fundamental adaptation to the ketogenic diet, seen in this specific cohort and similar groups of highly trained athletes, is to substantially increase whole-body rates of fat oxidation during exercise. This is achieved at the expense of an increased oxygen requirement (lower exercise economy); this is an outcome verified by other groups and explained by the stoichiometry of the pathways of fat and CHO oxidation. At lower exercise intensities (eg, <70% VO2 max), higher oxygen requirements for fat oxidation can be compensated for by increasing oxygen uptake (ie, working at a higher percentage of VO2 max) and may not impact performance. However, since exercise at or above lactate threshold (eg, interval/tempo training or elite endurance events) cannot sustain a further increase in oxygen uptake, exercise capacity is limited by a reduced ATP production from available oxygen supply or increased reliance on anaerobic energy production. This would account for both the reduction in speed in training sessions reported in the current data set, as well as the finding of reduced performance in the race that followed this study.

We note that our findings contradict observations from 2 other LCHF interventions: a 4-week study which found no apparent decrement in the capacity to complete a set high-intensity interval session at study mid- and endpoints or a 6-week study in which time to complete a 5-km treadmill run was reduced after 4 days but restored after 2 weeks. The reason for the discrepancies between these studies and our own data set is unclear, although it is noted that both investigations involved recreational/moderately trained individuals who provided their own dietary intake according to an education plan. Furthermore, neither group improved their performance over the 6 to 8 weeks training periods and minimal information on training characteristics was provided. It is possible that the participants in these studies achieved lower compliance to the intended diet and training intervention. Alternatively, the training completed by these lower tiered athletes might be considered a general exercise program with different characteristics and goals than the targeted and periodized programs undertaken by competitive athletes.

Although the training characteristics of the LCHF group reflected lower training volume and quality, they achieved an improvement in aerobic capacity over the 3-week period similar to the CHO-supported groups. The improvement in the ratio of the physiological (heart rate) and perceptual (RPE) response to a given (reduced) walking speed also shows that training under conditions of chronic low CHO availability is still able to provide a sufficient stimulus to provoke training adaptations. Indeed, on examination of the current data set, we suggest that the reported reduction in race performance following the training block is mostly explained by the acute effects of the limitations of fat-centric substrate utilization rather than an impaired training response. Of course, this observation applies only to the base phase of endurance training around which our study was designed and may not transfer to mesocycles targeting a competition peak in which there is greater ratio of high-intensity training sessions.

In support of our suggestion that the reduction in training characteristics was not a substantial contributor to the subsequent

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performance impairment in the current investigation, a subsequent and similarly designed study of our group found a performance decrement of similar magnitude following a 3.5-week adaptation to the LCHF diet. Here, the repeated finding of a ~7% differential in performance of a 10,000-m track race after the 3-week training block was explained by a 4.8% improvement in the group adhering to the high CHO availability diet and a 2.3% impairment in race time of the LCHF group. However, in this investigation, a subgroup of participants continued with 2.5 weeks of competition taper supported by a CHO-rich diet before competing in a 20 km race. Here, the improvement from baseline (measured in World Athletics [WA] points) was similar between CHO-supported (high CHO and periodized CHO) groups and LCHF athletes, showing that the previous decrement in performance could be overturned. Although some harmonization of performance outcomes might have also occurred as a result of the further training and taper integrated into the 2.5 weeks, this finding suggests that the acute restoration of CHO availability and utilization is important for the performance of high-intensity endurance exercise. We have also determined the importance of this latter aspect by a further finding that a 1-day CHO loading protocol was not able to restore CHO oxidation rates or overturn the performance decrement seen in athletes following an LCHF diet. Muscle retooling associated with adaptation to high-fat CHO restricted diets is associated with downregulation of glycogen breakdown and oxidation in tandem with the upregulation of fat storage, transport, and utilization in the muscle.

Within a training scenario, however, there are several options that might assist athletes following LCHF diets to rescue training quality during key sessions. Here, a case history involving an elite, long-term, keto-adapted, ultra-endurance athlete has reported that acute intake of CHO (60 g/h of training) prior to and during high-intensity sessions can improve performance of exercise associated with high rates of CHO oxidation without causing deadaptation. This might be partially explained by mouth sensing effects of CHO intake as well as small contributions to muscle fuel use. The use of caffeine during high-quality training sessions undertaken with low CHO availability may also help to partially restore training intensity and perceptions of effort. Although these strategies cannot not address the fundamental issues of impaired race performance associated with fat-centric substrate utilization, they may provide a “middle of the road” approach to enhance well-being and training quality during race preparation.

Here and as previously noted, adaptation to LCHF in elite athletes was associated with reduced perceptions of health and physical readiness, particularly in the first week. Transient reductions in well-being, often referred to as the “keto-flu,” potentially reflect the effects of reduced CHO availability prior to the increase in fat utilization and sustained ketone support for the CNS. Alternatively, reduced well-being could result from large electrolyte and fluid shifts occurring from the renal adjustment to the ketogenic diet, although our dietary control corrected for this as advised. Finally, the LCHF diet was associated with the development of an itchy dermatitis characterized as prurigo pigmentosa in 3 of the 10 athletes. Social media sites describe this as “the keto rash,” and although generally rare, dermatologists have noted an apparent increase in prevalence over the past 7 years, attributing this to the increased popularity of the LCHF diet. Although the etiology is unknown, it has been associated with other scenarios of ketosis including poorly controlled diabetes, fasting, anorexia nervosa, or bariatric surgery. A recent systematic review of 369 case studies included in 118 papers reported that of the 25% of patients who reported that dietary changes preceded the onset of the rash, 40% were associated with a ketogenic LCHF diet. Prurigo pigmento manifests as dense clusters of pruritic and hyperpigmented lesions, particularly on the chest and back. It is proposed that the high concentrations of blood ketones may lead to their accumulation around blood vessels, resulting in perivascular inflammation and neutrophilic infiltration. Treatment can include dietary changes or therapy with tetracyclines and other antibiotics. In the current cohort, the dermatitis quickly resolved on reintroduction of CHO-rich foods at the completion of the study. The high prevalence of prurigo pigmento has not been explained in our cohort, noting a previous association with ethnicity has been refuted and was not apparent in our group. Furthermore, we note that no cases have occurred in any of the other studies in which we have implemented similar LCHF diets in similar groups of athletes.

Practical Applications

This study showed adherence to a LCHF diet impaired training capacity, particularly training performed at high intensities. However, athletes who still wish to adopt a LCHF diet should consider potential modifications to their training plans to manage increased training stress and resulting fatigue, as well as selective CHO intake to support higher quality training.

Conclusion

In conclusion, elite endurance athletes adhering to an LCHF diet showed impaired training capacity relative to their CHO-supported counterparts, completing lower training volumes at slower speeds with higher heart rates. The increased exertion:velocity ratio suggests that internal load is still high and the training stimulus still allows exercise-induced physiological adaptations such as an increase in VO₂max to occur. However, the reduction in absolute training intensity may contribute to negative physiological (submaximal walking economy) and performance adaptations, especially during training phases that focus on high-intensity sessions.

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